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A geometric approach to the evolution of altruism

Andy Gardner

School of Biology, University of St Andrews, Dyers Brae, St Andrews KY16 9TH, United Kingdom

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ABSTRACT

Fisher's geometric model provides a powerful tool for making predictions about key properties of Darwinian adaptation. Here, I apply the geometric model to predict differences between the evolution of altruistic versus nonsocial phenotypes. I recover Kimura's prediction that probability of fixation is greater for mutations of intermediate size, but I find that the effect size that maximises probability of fixation is relatively small in the context of altruism and relatively large in the context of nonsocial phenotypes, and that the overall probability of fixation is lower for altruism and is higher for nonsocial phenotypes. Accordingly, the first selective substitution is expected to be smaller, and to take longer, in the context of the evolution of altruism. These results strengthen the justification for employing streamlined social evolutionary methodologies that assume adaptations are underpinned by many genes of small effect.

> *Finally, it must be pointed out that the model is not applicable to the selection of new mutations. Sibs might or might not carry the mutation depending on the point in the germ-line of the parent at which it had occurred, but for relatives in general a definite number of generations must pass before the coefficients give the true—or, under selection, the approximate—expectations of replicas. This point is favourable to the establishment of taking-traits and slightly against giving-traits.* — Hamilton (1964, p14)

1. Introduction

Fisher's (1930, pp38-41) geometric model provides a powerful tool for predicting key properties of the process and products of Darwinian adaptation (reviewed by Tenaillon 2014). Dispensing with the particulars of biology, it describes an abstract phenotypic space in which the fitness of any phenotype is a decreasing function of its Euclidean distance from the optimal phenotype, and in which mutations are represented by leaps of random magnitude in random directions within this phenotypic space. Fisher (1930) used the geometric model to argue that, on account of their higher probability of moving the phenotype closer to the optimum as opposed to taking it further away, mutations of small effect are more likely to be beneficial and hence contribute to adaptation. Kimura (1983, p155) subsequently pointed out that, conditional upon their being beneficial, mutations of larger effect are more likely to avoid stochastic loss, such that adaptation will proceed in steps of intermediate size.

In typical applications of the geometric model, the probability of fixation of a beneficial mutation is calculated upon the explicit or implicit assumption that the selection coefficient associated with the mutation remains constant over the course of its sojourn in the population. However, this assumption may be violated in the context of the evolution of altruism. Specifically, an allele encoding altruism may enjoy a selective advantage only insofar as the direct fitness cost experienced by its carrier is offset by benefits to other individuals who also carry copies of the same allele (Hamilton 1963). Accordingly, the allele will suffer a selective disadvantage upon its first appearance, in which it is present in only one copy, but may enjoy a selective advantage in subsequent generations in which it is present in multiple copies carried by multiple, socially-interacting relatives—provided that it has not already been lost from the population (Hamilton 1964, p14). The implications of this complexity for the geometry of adaptation remain to be investigated.

Here, I use the geometric model to predict differences in rate and genetic architecture for altruistic versus nonsocial adaptation. In an altruism scenario, I consider that fitness is modulated by interaction between clonemates; and in a nonsocial scenario, I consider that fitness is determined solely by the individual's own phenotype. I derive mathematical expressions describing probability of fixation as a function of mutational effect size under both altruism and nonsocial scenarios and use these to determine the average size of—and waiting time for—the next adaptive substitution, and I confirm these analytical results using numerical simulations.

E-mail address: andy.gardner@st-andrews.ac.uk.

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2. Model and analysis

Model—I assume a very large population of asexual, haploid organisms. In each generation a fixed number of haploid spores each give rise to two clonal individuals, and each individual gives rise to a Poisson-distributed number of spores, in proportion to her fitness. In the nonsocial scenario, I consider that an individual's fitness—i.e. expected number of offspring—is determined solely by her own phenotype and, in order that there be stabilizing selection about an intermediate optimum, I assume that fitness is given by $w = 1 + x - x^2$, where x is the individual's phenotypic trait value ($0 \leq x \leq 1$; Fig. 1a). In the altruism scenario, I consider than an individual's fitness is determined by both her own phenotype and also by the phenotype of her clonal twin, and is given by $w = 1 + y - x^2$, where x is her trait value ($0 \leq x \leq 1$) and y is the her twin's trait value ($0 \leq y \leq 1$; Fig. 1b); that is, the trait incurs a personal cost and provides a benefit to one's twin. Note that, insofar as twins are genetically identical, then they share the same phenotype ($y = x$), and hence the fitness functions are identical in both altruism and nonsocial scenarios. Only *de novo* mutation, arising in one twin and not the other, yields fitness differences between the two scenarios.

Geometry of adaptation—I consider a genetically uniform population in which every individual has trait value $x = 0$ (and, accordingly, every individual's twin has trait value $y = 0$), such that the average fitness of all individuals in the population is $\overline{w} = 1$. A mutational event is represented as one individual taking on a new trait value, uniformly distributed over the interval $0 \leq x \leq 1$, which then either proceeds to fixation under the action of natural selection or else is lost from the population.

The mutant allele's probability of fixation depends upon its selection coefficient. In the nonsocial scenario, an initial mutation of size x results in fitness $w = 1 + x - x^2$, and hence a relative fitness of $1 + s$ where $s = x - x^2$, consistently in every generation. In the altruism scenario, an initial mutation of size x results in fitness $w = 1 - x^2$ in its first generation (owing to the carrier individual's twin having wildtype trait value $y = 0$), and it enjoys fitness $w = 1 + x - x^2$ in every subsequent generation (owing to the twin also carrying this allele, such that $y = x$). That is, in the altruism scenario, the mutation of size x results in relative fitness $1 - t$ in the first generation and relative fitness $1 + s$ in every generation thereafter, where $t = x^2$ and $s = x - x^2$. In the Appendix, I derive expressions for the probability of fixation $p_N(x)$ of a mutation of effect size x in the nonsocial scenario, and for the probability of fixation $p_A(x)$ of a mutation of effect size x in the altruism scenario. Solutions for these fixation probabilities are given in Fig. 2a, along with results from numerical simulations (see Appendix for details).

The overall probability of fixation for a mutation whose random, uniformly distributed effect size falls within the unit interval, is $\pi_N = \int_0^1 p_N(x)dx = 0.143$ for the nonsocial scenario and $\pi_A = \int_0^1 p_A(x)dx = 0.086$ for the altruism scenario (these values correspond to the areas under the curves in Fig. 2a). That is, without conditioning on a mutation's effect size, its probability of fixation in the altruism scenario is, on average, only 60% of the probability of fixation of the corresponding mutation in the nonsocial scenario. Accordingly, the evolution of altruism is expected to proceed more slowly than nonsocial adaptation, with the expected waiting time until the appearance of the first successfully fixing mutation being 66% longer (Fig. 2b).

This impeding of the evolution of altruism is stronger for mutations of larger effect, which skews the distribution of effect sizes of successfully fixing mutations in favour of smaller values. The probability distribution of effect sizes of the first successfully fixing mutation is given by $\phi_N(x) = p_N(x)/\pi_N$ for the nonsocial scenario and by $\phi_A(x) = p_A(x)/\pi_A$ for the altruism scenario. These distributions are given in Fig. 2c, along with results from numerical simulations (see Appendix for details). Finally, the expected effect size of the first fixed mutation is $x^*_N = \int_0^1 \phi_N(x)xdx = 0.500$ for the nonsocial scenario and $x^*_A = \int_0^1 \phi_A(x)xdx = 0.425$ for the altruism scenario (Fig. 2c). That is, the expected size of the first fixed mutation in the altruism scenario is only 85% of the expected size of the first fixed mutation in the nonsocial scenario.

3. Discussion

Using analytical and numerical simulation methods, I have shown that the evolution of altruism is expected to proceed in smaller steps, each taken more slowly, than is nonsocial adaptation. These results owe to a basic asymmetry in probability of fixation. In a nonsocial setting, a mutant allele that would be expected to rise towards eventual fixation in a purely deterministic world has some probability of being lost from the population soon after its first appearance, and this probability is reduced if the mutation has a larger beneficial effect, on account of natural selection working more effectively to counter its stochastic loss. In contrast, in the context of altruism, a similar mutant allele may actually be disfavoured by natural selection upon its first appearance if its long-term advantage owes to benefits it provides to its carrier's genealogical kin—as initially these kin will not carry copies of the same allele—and hence its probability of stochastic loss in its first generation of existence is reduced if it has a smaller phenotypic effect.

The asymmetry between altruistic versus nonsocial adaptation is not expected to affect the phenotype that is eventually reached at the end of the adaptive trajectory, but it is expected to impact upon this trait's genetic architecture, with altruism being predicted to be underpinned by a relatively larger number of genes with relatively smaller phenotypic effects. Such quantitative genetic architecture is a key assumption for a range of theoretical evolutionary methodologies, including the Taylor-

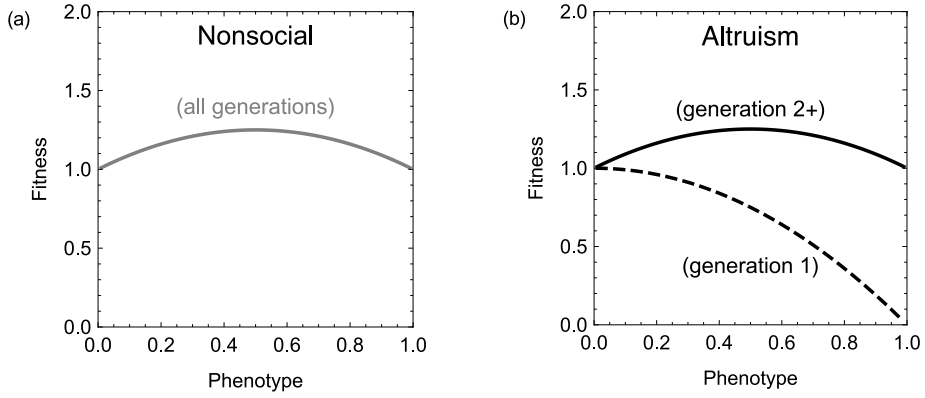

Fig. 1. The fitness landscape. (a) In the nonsocial scenario, the fitness of a focal individual bearing a mutant allele is given by $w = 1 + x - x^2$, where x is the phenotype encoded by the mutant allele. (b) In the altruism scenario, fitness of a focal individual bearing a mutant allele is given by $w = 1 + y - x^2$, where x is the phenotype encoded by the mutant allele, and where the clonal partner's phenotype is given by $y = 0$ (dashed line) upon the first appearance of the mutant allele and by $y = x$ (solid line) in every subsequent generation.

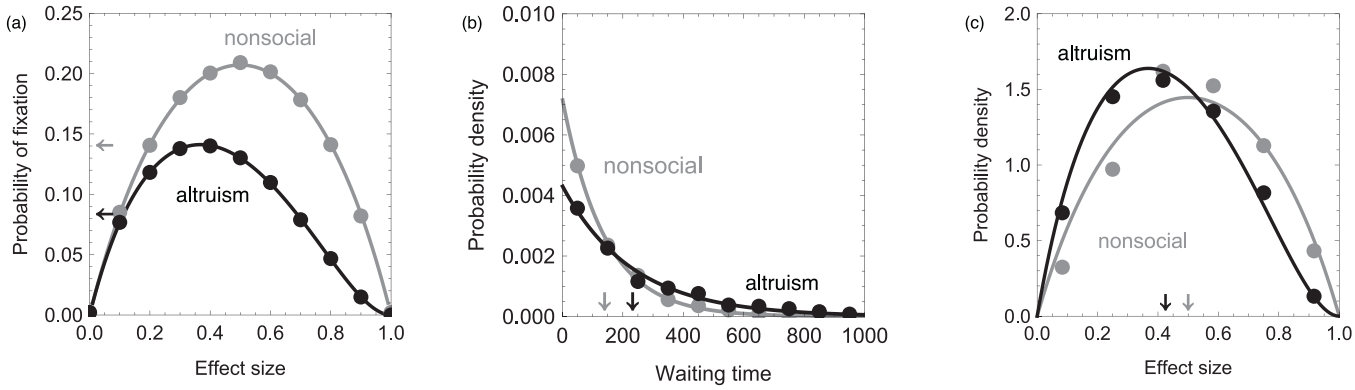

Fig. 2. Probability of fixation of mutant alleles (panel a), distribution of number of generations until the appearance of the first successfully fixing mutant allele (panel b), and distribution of effect size of the first successfully fixing mutant allele (panel c), in nonsocial (grey) and altruism (black) scenarios, derived analytically (lines) and confirmed by numerical simulation (dots). Arrows indicate analytically derived averages for the nonsocial (grey) and altruism (black) scenarios.

Frank neighbour-modulated fitness approach to modelling kin selection (Taylor & Frank 1996), in which the assumption of vanishingly small amounts of genetic variation enables fitness-trait regressions to be captured as derivatives of fitness functions, facilitating the use of powerful calculus techniques that help to render models more analytically tractable. The present analysis suggests that this assumption has relatively better justification in the context of the evolution of altruism.

The aim of the present analysis has been to provide a quantitative illustration of this basic asymmetry between altruistic versus nonsocial adaptation, rather than a fully general account. I have assumed a highly stylized population in which mutations are considered to arise in such a way that they are never shared by social partners in their first generation of existence and are always shared by social partners in all subsequent generations. More generally, depending upon its precise timing, a new mutation could initially be carried by multiple siblings, and insofar as social interaction occurs between more distant relatives it might take multiple generations for relatedness to attain its equilibrium value (Hamilton 1964). With regard to the latter, Mullon & Lehmann (2014) have reported a similar impediment to altruism in an island-model setting that occurs in all generations of an allele's sojourn, rather than only the first generation. In contrast to the present effect, which owes to genealogical relatives initially being genetically unrelated, theirs owes to dispersing altruists being unrelated—both genetically and genealogically—to social partners and hence struggling against strong selection to establish their lineage locally.

Moreover, the present model has assumed haploid inheritance, such that the complexities of dominance and recessiveness do not arise. In a diploid setting, recessive alleles encoding altruism would be invisible to selection while present in only one copy upon their first appearance in the population, and hence would be less susceptible to immediate loss. This mirrors "Haldane's sieve", whereby dominance facilitates the fixation of (nonsocial) beneficial alleles on account of recessive beneficial alleles being more liable to stochastic loss while present in only a small number of copies (Haldane 1922, 1924; Turner 1981). Furthermore, although the geometric model focuses attention on the supplanting of a wildtype by a *de novo* mutant phenotype, adaptation to new selection pressures will usually be fuelled, at least in part, by standing variation in respect to which genealogical kin are already genetically correlated.

To facilitate comparison, the present analysis has considered altruism versus nonsocial scenarios that are contrived to be identical in every respect other the core asymmetry of interest, in order that this obstacle to the evolution of altruism may be carefully isolated and quantified. More realistically, in any comparison between a given altruism phenotype and a given nonsocial phenotype, many additional factors are liable to be confounding. Microbes might be relatively amenable to an experimental test involving, for example, a contrast between antibiotic-resistance conferring enzymes with intracellular (private good) versus intercellular (public good) activity (Frost et al. 2018)—representing nonsocial versus altruistic scenarios, respectively—and more readily allowing for whole-genome evolution to be tracked over multiple generations.

The present analysis focuses upon a particular social scenario in which the individual has full control over her phenotype and where her phenotype modulates both her and her social partner's fitness. Mutational substitutions of larger effect might be expected in other social settings, including in the context of evolutionary conflicts in which different parties, with different fitness interests, share control over the phenotype (Scott & Queller 2019; Rautiala & Gardner 2023). Indeed, Rautiala & Gardner's (2023) application of the geometric approach to conflict scenarios has highlighted the possibility for successive fixed mutations to "leap-frog" from the far side of one conflicting party's fitness optimum to the far side of an opponent's, and back again, resulting in an arbitrarily large degree of maladaptation and starkly contrasting with the monotonic increase in fitness that characterizes Fisher's (1930) original model.

Declaration of Competing Interest

The author declares that he has no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix

Fixation probability—An allele that confers a consistent relative fitness advantage s in every generation ultimately fixes in the population with probability

$$p = 1 - \sum_{k=0}^{\infty} \frac{e^{-\frac{1+s}{2}}\left(\frac{1+s}{2}\right)^{k}}{k!}(1-p)^{2k} = 1 - e^{-\left(\frac{1+s}{2}\right)\left(1-(1-p)^{2}\right)} \tag{A1}$$

conditional upon initially being present as a single copy. That is, its carrier produces a Poisson-distributed number k of spores, with expectation $(1 + s)/2$, which yield $2k$ offspring, of which each has probability $1\text{-}p$ of leaving no descendants in the long term; the probability that the carrier herself leaves no descendants in the long term is given by the probability that none of her $2k$ offspring do so, and the probability that this outcome does not obtain defines the probability of eventual fixation. It is straightforward to show that $p \approx s$ for small s; note that this differs from Haldane's (1927, p839) result $p \approx 2s$, by a factor of two, on account of the clonal duplication of spores in the present model, which is done as a convenient means for implementing social interaction between relatives. More generally, an explicit analytical solution to this equation is not possible, but the equation is readily solved numerically. In relation to the nonsocial scenario described in the main text, the fixation probability of a mutation of effect size x is given by $p_{\mathrm{N}}(x) = p|_{s=x\text{-}x\hat{}2}$.

An allele that confers a relative fitness disadvantage t in its first generation and a consistent relative fitness advantage s in all subsequent generations ultimately fixes in the population with probability

$$p' = 1 - \sum_{k=0}^{\infty} \frac{e^{-\frac{1-t}{2}}\left(\frac{1-t}{2}\right)^{k}}{k!}(1-p)^{2k} = 1 - e^{-\left(\frac{1-t}{2}\right)\left(1-(1-p)^{2}\right)} \tag{A2}$$

where p is given by equation (A1), as before. That is, only the probability distribution of the initial carrier's number of offspring differs between the altruism and nonsocial scenarios, owing to the single-generation disadvantage accrued in the altruism scenario; conditional upon its initial carrier producing a given number of offspring $2k$, the allele's evolutionary prospects are the same in both scenarios. Again, an explicit analytical solution to this equation is not possible, but the equation is readily solved numerically. In relation to the altruism scenario described in the main text, the fixation probability of a mutation of effect size x is given by $p_{\mathrm{A}}(x) = p'|_{s=x\text{-}x\hat{}2,t=x\hat{}2}$.

Numerical simulations—To confirm the fixation-probability results, a numerical simulation is conducted in which a population of 250 spores undergoes doubling to produce 500 individuals, each of which then produces a large number of clonal spores in proportion to its fitness (as described in the main text), of which 250 spores are chosen at random to form the next generation. In the first generation only, one individual is assigned a mutant phenotypic value of x, and all other individuals are assigned phenotypic value of zero. 10^3 generations of evolution are followed, after which the mutant is said to have become fixed in the population if it is still present and to have been lost from the population if it is no longer present; this approach potentially overestimates the probability of fixation, as beneficial alleles that have persisted for 10^3 generations might subsequently be lost from the population, but in practice it provides a good approximation. 10^5 replicates are performed, yielding an estimate of the fixation probability. This procedure is repeated for several different values of x and for both altruism and social scenarios. *Mathematica* code used to run the simulations and generate Fig. 2a is provided as supplementary material.

To confirm the distribution-of-waiting-time and effect-size results, a numerical simulation is conducted in which a population of 250 spores undergoes doubling to produce 500 individuals, each of which then produces a large number of clonal spores in proportion to its fitness (as described in the main text), of which 250 spores are chosen at random to form the next generation. Initially, all individuals have a phenotypic value of zero. In every generation, every individual has an independent probability 10^{-4} of being assigned a mutant phenotypic value that is uniformly distributed over the unit interval, and the generation in which this mutation arises is recorded. If a subsequent mutational event occurs in an individual already holding a mutant phenotype, the individual is considered to retain the original mutation and to also accrue a new mutation elsewhere in their genome. 2×10^3 generations of evolution are followed, after which one individual is chosen at random and the earliest-arising mutation in its genome is examined. The generation in which this mutation first appeared and the size of the mutation is recorded. 10^3 replicates are performed, yielding distributions for both waiting time and effect size. This procedure is then repeated for both altruism and social scenarios. *Mathematica* code used to run the simulations and generate Fig. 2b and c is provided as supplementary material.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jtbi.2023.111653.

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